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THE NEWER DERMATOLOGICAL HAZARDS OF INDUSTRY.*

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THE Section of Dermatology of the Royal Society of Medicine has for long shown a sustained interest in the dermatological aspects of industrial medicine—an interest which has extended beyond the giving of opinions in consulting room or hospital, and manifest by an enthusiasm to see working conditions as they exist in the factory and thus to determine and evaluate at first hand, the many factors at play in the production of dermatoses there.

The man or woman at work has another life outside the factory at home, in the garden, in the workshop or garage, and it is hard indeed in practice to live up to the axiom of “considering man as a whole”. We cannot know too much about him and his personal history if we are to crack the nut of “idiosyncrasy” and “special susceptibility”—the dim curtain of obscurity which hangs between us and the truth.

The French say that living, as we are thought to do, in a dense fog for so many months in a year, we make good investigators. Perhaps one skill we have is in recognizing where other investigators have gone wrong. Recently in the United States a study on a very large scale, of dermatitis in industry with special reference to oils as a causative factor, relied, for incidence of cases, on those workers who reported their condition to the works surgery.

There are two major points here—the first is that if you want to find out what is the real incidence of skin damage, and especially the early signs of this, in a factory, you must emulate the example of the woman in the parable, take a candle, sweep the floor and search diligently.

* Based on the opening address of a symposium on New Industrial Dermatological Hazards held by the Section of Dermatology of the Royal Society of Medicine on 19 March, 1959.

But there is a vital difference—for in the search in the factory you must not determine narrowly beforehand what you are looking for.

The second point of tremendous importance is to study with equal care the people whose skins appear to withstand the industrial challenge, while others similarly employed have fallen by the way.

It may be that for some work the "normal" reaction is towards dermatoses, or at least some skin damage, whilst this abnormal skin which escapes injury is overlooked.

The "newer" dermatological hazards of industry are often those of yesterday, and the day before yesterday, but in new guises associated with the more modern processes and methods of work. What MacKenna wrote of *acne vulgaris*, "the eruption is due to malevolent synthesis between fundamental and subsidiary factors" is true of many occupational dermatoses.

The skin hazards of adhesives have been for many years recognized—they threatened essential production in the war years. Now we have them in an aggravated form as the newer synthetic resins, such as the epoxy resins—polyethers with a relatively short chain with hydroxyl groups at regular intervals, and an epoxy group at each end. Epichlorhydrin, well known as an eye irritant at high temperatures, plays a part in their formation, as does caustic soda, while acid anhydrides come in later as curing agents.

It is essentially the curing or hardening of the epoxide groups by various chemicals, particularly the polyamines, which has caused so much trouble, for it introduced an at first unrecognized fume hazard to add to those of skin contact with the resin during mixing, throughout the curing stage and perhaps afterwards.

The amines are well known in some hair dyes and as accelerators and anti-oxidants in the rubber industry (synthetic rubber manufacture uses more than are required for the natural product) while in the dyestuffs industry and elsewhere we have a profound respect amounting to aversion for β -naphthylamine, which is no longer manufactured here, because of its carcinogenic effect on the urinary tract, especially the bladder.

The particular polyamines used for epoxy resin curing (they are the most versatile of these agents) are strongly alkaline, fume in air, and they have an irritant and even corrosive effect on body tissues. They are also potent sensitizers.

This is an older dermatological hazard in new clothing as is that of fibreglass now used sometimes with the epoxy and more generally with the polyester resins for processes including the making of fabricated car bodies.

Chromium compounds crop up in new places such as for preserving sample milk in the Netherlands, and for addition to water used for blasting materials to remove rust. Mineral oil whether "straight" or emulsified has divers

additives for functional purposes—some of these may be responsible for inflammatory reactions of the skin which are so unlike oil acne.

Insecticides are made in factories but their uses spread beyond agriculture and horticulture and they are increasingly found in factories. The hazards to health may be far more than skin reactions and absorption through the skin is an important mode of entry to the body.

Organic tin compounds are of increasing importance in industry, as stabilizers in plastics and polymerizers in silicone manufacture. They are also effective as fungicides in paper making, paint manufacture and in the preservation of timber. Two kinds of eruption have been observed, the acute local burn and a subacute dermatitis on areas where contaminated clothing has rubbed against the skin on the trunk and thighs and on the calves level with the tops of Wellington boots.

“Black fingers” of girls on soldering has been traced to fluoride in the flux.

Of interest today are the reports of beryllium granuloma and even of a granuloma attributed to zirconium in a toilet preparation. Both these metals are in contemporary demand because of the heat they will withstand—as in atomic reactors.

The occasional case of lung sarcoidosis in industry is puzzling and the dermatological manifestations may throw light on the etiology of this condition which does not seem excessively rare.

In spite of an intense drive to ensure protection for workers against ionizing radiations in its many forms, there occur a few cases of skin damage from beta, X- and gamma-radiation. There is in draft a Code of Regulations to control the use in establishments under the Factories Act of sealed sources of ionizing radiations including X-rays, while there is in evolution an appropriate Code for unsealed sources.

There are other skin carcinogenic agents in industry. Elimination of the skin carcinogenic types of mineral oil in mule spinning by the 1953 Code of Regulations is fundamentally sound and it should in course of time eradicate mule spinner's cancer. Meantime, statutory periodic medical examination of workers, many of whom have had exposure to potentially carcinogenic spindle oils, should ensure early diagnosis and treatment; those first employed since 1953 are being watched to see if the substitution of white oils has proved successful.

Cortico-steroids are increasingly used in the treatment of systemic disease as well as for topical application. How these preparations, as well as systemic and local antibiotic treatment, affect the ultimate resistance of the skin to the often subtle attack of external irritants, is not as yet assessable.

Now is available information affording more knowledge than is yielded by voluntary notification of the incidence of dermatitis in that part of the insured

population of these Islands who work in factories. There are also more Medical Inspectors to study at first hand in the factories the causes and prevention of dermatitis and industrial skin cancer. Industrial medical officers and nurses in industry give special attention to these questions.

In the prevention of pathological skin conditions in industry there is an increasing awareness of the importance of the working environment in maintaining the skin in a state of health. Safeguards external to the worker are fundamental to this end. There is a fuller estimation of what protection can be given to a worker's skin by such things as gloves, overalls, and boots, and of what a "barrier" can, or cannot, be expected to achieve as a part of the whole scheme of protection.

Great care is taken in the preparation of official memoranda and placards for the education of both sides of industry. The placards are intended to be essential information to the often-forgotten man at risk on the factory floor telling him too how he can avoid running into trouble and what to do if he does. Inevitably, these publications are referred to in the setting of legal standards of good practice, primarily looked for from the employer. Sometimes the result of a Common Law action hangs on whether or not a substance is specifically included among the examples of skin irritants listed in one or other of these publications, while the official advice on barrier preparations may tip the balance.

More and more credit is being given to the value of thorough removal by harmless cleansing processes, of harmful materials if these come in contact with the skin, as they must do inevitably in some work, in spite of measures aimed at minimal or no skin contact. The health of the skin depends on the maintenance of its *status quo*—and this maxim for all its brevity is not unmindful of the complex biochemical and physical issues involved.

There is something at once frightening and stimulating in the questions of Pythagoras—

“What have I learnt where'er I've been

From all I've heard, from all I've seen ?

What know I more that's worth the knowing ? ”

Gratitude is due to all dermatologists who concern themselves with what their patients do in factories and especially to dermatologists who give their advice to those who are in some measure responsible for the health of the industrial population.

THE EFFECTS ON THE SKIN OF SOME NEW ADHESIVES AND LAMINATING MATERIALS.*

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ADHESIVES were formerly based on skin, bone, fish or starch. Hazards included fire risks, traumatic dermatitis from glue hardening on the skin or abrasive spicules (e.g. silicates), the alkali effect of the casein glues and occasional sensitization by the glues themselves, or by preservatives such as formaldehyde and pentachlorophenol.

Adhesives are now produced resistant to heat, solvents, water, chemicals and bacteria, and which can make firm joints with such diverse materials as fabrics, rubber, wood, glass, ceramics, metal or plastics. In some, additional risks of skin irritation may occur. Here are considered rubbers and synthetic resins of the formaldehyde, polyester, isocyanate, silicone and epoxide type, as used for adhesives or lamination.

RESIN CHEMISTRY.

Kienle first indicated the principles underlying synthetic resin and polymer formation (Martin, 1951). For a reaction to occur either by condensation of two dissimilar molecules, or by polymerization of two like molecules (a monomer) each must have more than one functional reactive group. Common functional groups in adhesives are hydroxyl (OH) carboxyl (COOH) amine (NH₂) epoxy $\begin{array}{c} \text{O} \\ \diagup \quad \diagdown \\ (\text{CH}-\text{CH}_2) \end{array}$ isocyanate (NCO) or unsaturated double bonds. If each molecule has two reactive groups then a long chain thermoplastic polymer is formed. If one of them has more than two groups, then a third unsatisfied group is free to form a cross link between the individual long chains to form a completely polymerized thermosetting macro-molecule.

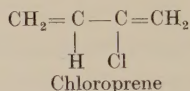
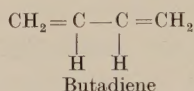
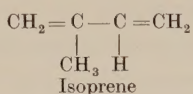
Synthetic adhesives may be regarded as partially polymerized long-chain molecules which, on the addition of a catalyst or hardener, with heat and light, cross link between remaining functional groups to an infusible inert chemical state. After the hardener or catalyst is added, the time before curing occurs is called the "pot life". Completely polymerized and cured (cross linked) resins are chemically inert and are not skin irritants or sensitizers, but

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initial condensation products, monomers or intermediate products can be irritants or sensitizers, as can some of the curing or cross linking agents. When a cured resin gives rise to dermatitis it probably does so from incomplete cure or persisting intermediates.

RUBBER ADHESIVES.

Natural rubber is a giant polymer made up of chains of isoprene molecules with unsaturated double-bond linkages. Synthetic rubbers derive from similar unsaturated compounds such as butadiene, chloroprene or co-polymers of butadiene, styrene or isobutylene.



All rubbers are used in solid form or as milky dispersions (latex) which are made alkaline (pH 8-10) for preservation. Cure or vulcanization occurs by cross linkages using sulphur or such related chemicals as selenium or tellurium. In order to impart various properties to the final rubber, the compounding mix includes many chemicals including anti-oxidants, plasticizers, fillers, dyes and special additives, in addition to activators, accelerators and retarders which alter the speed of this normally slow process of vulcanization. Many of these added chemicals are accepted as skin irritants or sensitizers.

Rubber adhesive solutions may or may not require vulcanization. They are of three types, although in many proprietary glues they may be mixed with other resins or adhesives.

(i) *Non-vulcanizing Rubber Solutions.*

These contain natural or synthetic rubber in a petroleum solvent such as naphtha, benzene, petrol or xylol with wood rosin, ester gum or shellac for tackiness. They are used in the shoe industry, for surgical tape or for motor car trimmings. Hazards can mainly arise from the solvent or the resins.

(ii) *Vulcanizing Solutions or Cements.*

These contain compounded natural or synthetic rubber with fillers, adhesive agents and vulcanization accelerators (e.g. zinc dithiocarbamate, zinc isopropyl xanthate or mercaptobenzothiazole). They may be supplied as two solutions for mixing together before use, with a "pot life" of some hours. They are used for joining rubber, mending punctures etc. Sensitization dermatitis may occur from the compounding rubber chemicals.

(iii) *Latex Cements.*

These are made of latex rubber with or without solvent naphtha, with wetting agents, glue, ester gum and disinfectants. They are used in the shoe and

textile trade. Dermatitis may result from the alkaline effect of ammonia or the solvent effect of disinfectants. The shoe industry also employs iron—in adhesives for cloth impregnated with gutta percha, also fish glues, pyroxylin-cellulose acetate and cellulose nitrate cements or synthetic resins such as polyvinyl acetate and polyisobutylene. Almost any operation in the shoe trade may now be done with adhesives.

Other uses for rubber include preserved latex with compounding agents mixed with cement or gypsum for filling in brick work. For paper leather-cloth, one side is coated with the compounded rubber solution but no sulphur, the other with the solution and no accelerator, vulcanization and fusion occurring when they come together.

FORMALDEHYDE RESINS.

These are used as adhesives in woodworking, e.g. furniture, aircraft, and plywood manufacture; lamination of paper, cotton or fabrics by impregnation and pressure moulding; hard board manufacture from paper pulp or wood chips and in foundry work for sand shell moulding.

Phenol-urea-melamine or resorcinol-formaldehyde resins are employed. Cure takes place by heat alone (as with phenol-formaldehyde resins in veneers of plywood pressed together by heat), or by the addition of a catalyst or hardener to cross link the chains, or which alters the pH, allowing cross linkage. Urea and resinol resins set at acid pH. The resins are sold for adhesives as solids, aqueous syrups or dissolved in methylated spirit (methanol).

They all contain free formaldehyde, polymerization having been stopped at various stages short of full cure, e.g. urea syrups, 3–4 %; resorcinol, less than 1 %; phenol, 3–4 % and melamine, 3–4 % free formaldehyde. Hardeners and catalysts include ammonium chloride (which with the free formaldehyde in urea resins forms hexamine), paraformaldehyde and hydrochloric acid, urea and hexamine itself.

In one successful system for bonding metal, wood, plastic or rubber together, a liquid phenol-formaldehyde in methylated spirit is applied followed by a polyvinyl formol powder. All these adhesives have been in constant use for many years and when occasional dermatitis occurs this is usually caused by formaldehyde (or its vapour in heat set processes) or by the hardeners.

It is thought that trauma from the adhesive on the skin is the primary injury, the formaldehyde sensitization being secondary (MacKenna and Horner, 1954). Para-tertiary butyl phenol used with a phenol formaldehyde adhesive resin has been reported as a sensitizer (Malten, 1958). Most exposure occurs in batching, glueing, machining and finishing, or by contamination of clothes when washing out the glue receptacles.

Preventive measures are outlined in H.M.S.O. Factory Department, Ministry of Labour and National Service form No. 331, Feb. 1943, reprinted 1956.

POLYESTERS.

These are made by condensation of a di- or polyhydric alcohol (propylene, diethylene or ethylene glycol) with a di- or polybasic organic acid or anhydride (maleic, phthalic or fumaric). The straight chain polymer has unsaturated double-bond links and is liquid or solid, according to the length of the chain.

Cure occurs at, or about, room temperature by using an unsaturated monomer such as styrene (vinyl benzene) $C_6H_5CH = CH_2$ or methyl methacrylate, which polymerizes and cross links the chains at the double bonds. This is assisted by an oxidizing catalyst, e.g. benzoyl peroxide, tertiary butyl hydroperoxide, or methyl ethyl ketone peroxide (MEK) with cobalt naphthenate as an accelerator. Cobalt salts, incidentally, are used as driers in paints and as such have been reported as sensitizers by Pirilä, 1947.

The polyester is supplied for use as a mixture of polyester 70% and styrene 30% with traces of hydroquinone or tertiary butyl catechol to inhibit oxidation which might cause premature polymerization in storage. The oxidation catalyst is added before use.

The system is used for bonding cloth, glass fibre or asbestos into laminates for car bodies or boats, for mouldings like corrugated sheeting, pit props, firemen's helmets, or tennis racquets and also as a plasticizer for polyvinyl chloride resins in food packaging, adhesive tapes, artificial leather, etc.

Dermatitis may arise from the following possibilities.

(i) Styrene has an irritant and solvent effect like benzene; it is also a fire risk.

(ii) The polyester resin is probably safe but sensitization can occur (Malten, 1956 and 1957) from fumaric acid and esters of maleic acid (diethyl) in the resins. Sensitivity to simple phthalate esters is not recorded. According to Dani and Mitchell (1956), continued cross linkage can go on for several weeks after storing and near completion of curing. Machining of such laminates could theoretically release intermediate products.

(iii) Acetone is used as a solvent for the resin and for hand cleaning; it can be an irritant.

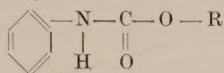
(iv) Other risks are the potential irritant effects of catechol (phenol-like effect) and hydroquinone inhibitors and the irritant, explosive and fire risks of the oxidizing agents, methyl ethyl ketone peroxide and benzoyl peroxide.

(v) The fingers of protective rubber gloves adhere together after contact with the resin; polyvinyl chloride gloves are not affected.

POLYURETHANE RESINS (ISOCYANATES).

An isocyanate ($-NCO$ reactive group), usually toluene di-isocyanate, reacts with the hydroxyl $-OH$ group of a polyalcohol (glycol). A prepolymer is formed which later reacts with more toluene di-isocyanate to form a long chain

polymer linked by urethane groups



During the reaction, freed carbon dioxide gas becomes trapped in the syrupy resin and makes a foam. The polymers can be cross linked with the polyester system to a thermosetting form. These cellular and foamed plastics are moulded and used for upholstery and packaging, for sound and heat insulation or as adhesives.

In actual use, the prepolymer, the di-isocyanate, the polyester catalyst and water are mixed together and poured into a mould or sprayed on a surface in successive layers as a lacquer. A solution of polyisocyanate in *xylene* may be used for bonding rubber to textiles, metals or ceramics.

The final polymerized foam plastic gave no positive patch tests in a series (Zapp, 1957) but toluene and other di-isocyanates are primary skin irritants. They are also eye, nose and throat irritants and there are reports of their inducing asthma in susceptible persons (Swensson *et al.*, 1955). The polyisocyanate monomers have a low index of skin irritant and sensitization effect. Some possibility of contact with vapour can occur during the foaming reaction.

GLASS FIBRE.

As glass fibre laminates and engineering castings are commonly bonded with polyester, epoxide or occasionally silicone resins, this will be briefly considered.

The glass fibre is made by feeding the raw materials or composite glass marbles into an electrically heated furnace whence the molten glass flows out of small holes and is drawn on to a revolving drum as a continuous filament, or is shattered by steam blast to a cotton wool form. Continuous filament is wound to yarn or rope or chopped to a needle mat. It is woven to a cloth or tape for textiles or reinforced laminates and later bonded by adhesive resins. A coating lubricant emulsion is sprayed on to the filament as it is drawn from the furnace. Where the fibres are to be bonded by formaldehyde, polyester, epoxy or silicone resins, intermediate or keying resins are added to the emulsion to get firm adhesion between the glass and these bonding resins. Vinyl trichlorosilane (Volan) and methacrylate-chromyl chloride are used as keying agents.

Formaldehyde resin bonded boards for acoustic ceilings, refrigerators, cookers, or moulded pipe sections are made by spraying the glass wool with the resin and passing it through an oven for curing, later cutting to size. There is probably no formaldehyde risk to the user ; only a minimal amount of uncured resin remains. For roof insulation, the carpet of glass wool is sprayed with aqueous bitumen emulsified with ammonium caseinate and rolled up for use.

The main hazard of glass fibre is penetration of the skin by small spicules of glass. Sensitization by glass fibre is not recorded (Heisal and Mitchell, 1957). The commonest eruption is the "new starters" rash after 2-3 days' contact. This is a measles-like eruption of tiny papules on exposed areas and in skin creases. The eruption clears with "hardening" in about 2-3 weeks. Other lesions include purpura, telangiectasia, folliculitis, urticaria, linear erosions in skin creases, paronychia from spicules under the nail fold, frictional eczematous dermatitis from impregnated clothing, irritation of other members of the family at home, sepsis and pulp infections. Workers in the actual manufacture may develop a pustular folliculitis like oil acne from hydrogenated vegetable oils and the organic chemicals of the coating emulsion; also eczematous dermatitis from the heat and moist conditions with burns from hot slugs of glass and a non-mycotic maceration of the feet from the coating of leather footwear with glass fibre and resin sufficient to interfere with its porosity (McKenna, Ferguson Smith and MacLean, 1957).

SILICONES.

Silicones are straight chain polymers with linked silicon and oxygen atoms.

Their viscosity increases with the chain length and this and the differing side chains determine their industrial uses. There are short chain oils for polishes, hydraulic brake fluids or mould release agents, longer chain oils for water repellant textile emulsions, paint or glass fibre electrical laminates, greases for lubrication and finally gum-like rubbers which are sold with the oxidizing benzoyl peroxide already incorporated, whose cure results from heat alone.

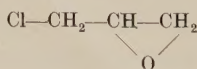
None of the silicones is a skin irritant. In the manufacture the action of methyl chloride on silicon produces crude silanes which later hydrolyse to silanols and thence to silicones. The silanes are skin irritants yielding hydrochloric acid on hydrolysis, and on skin contact cause dehydration and acid burns.

EPOXY RESINS.

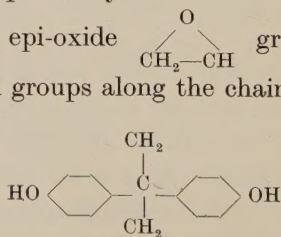
Ethoxylines or epoxides; Epikote, Shell; Araldite, Ciba; Epon, U.S.A.

Epoxy resins are formed by condensation of a polyhydric phenol (diphenylol propane or isopropylidenediphenol) with epichlorhydrin.

The long chain polymer has reactive epi-oxide groups at each end and intermediate hydroxyl functional groups along the chain.

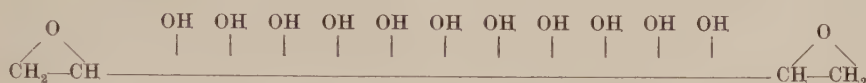


Epichlorhydrin

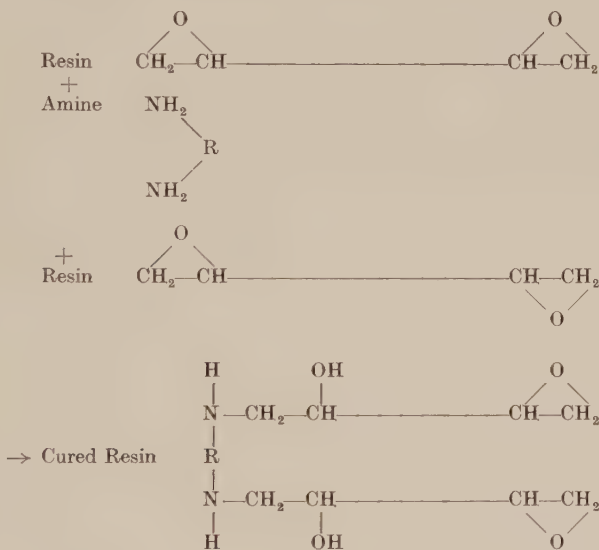


Diphenylol propane

The polymer may be represented diagrammatically as



Industrial uses depend upon reacting these functional groups with other chemicals. The hydroxyl group allows ester formation with fatty acid radicles for air drying paints or with phenol formaldehyde or alkyd resins for stoving paints, which dry on heating. Reaction of the terminal epoxide group with a curing agent or hardener, which is commonly an amine, breaks the epoxide ring and cross links the individual molecular chains to a tough infusible and chemically inert resin.



Any further epoxide chains can react with the hydrogen atoms until a complete lattice or sponge work is built up with cross linkage in all planes.

This epoxide-amine curing system is used for adhesion to wood, glass, ceramics, plastic or metal—it may indeed be used instead of welding—also for glass fibre laminates for aircraft or motor car bodies and for engineering casting, or the potting and encapsulation of electrical components and for chemical-resistant paints. In castings, the final resin is further altered by fillers (e.g. sand, finely powdered silica, alumina, iron powder, etc.) and modifying resins such as polysulphide polymers (Thiokol) or polyamides.

Epoxide resins are liquid or solid according to the length of the polymer chain. Of the initial reactants, epichlorhydrin is a powerful skin irritant. As sold by the makers, the composition of the epoxide resins may vary considerably. The resin for instance may contain in addition (i) solvent diluents,

(methyl ethyl ketone—Butanone—cellosolve, phenyl glycidyl ether, alkyd glycidyl ether, toluene, xylene, styrene, acetonitrile), (ii) plasticizers for softening (dibutyl phthallate or tricresyl phosphate), (iii) epoxide resin modifiers (a straight chain chemical with terminal epoxide groups) or (iv) small quantities of unreacted epichlorhydrin. The curing agents are primary, secondary or polyamines, amine salts or anhydrides. Ethylene amines are frequently used. These amines may be supplied in a solvent solution. Although cure takes place at room temperature, the process being exothermic, it is speeded up by moderate heat or added accelerators such as catechol. Some amines decompose at high temperature and give off irritating vapours.

The epoxy resin and the hardener is called the epoxide-amine system. Before use, the polyamine hardener is mixed with the resin. In actual practice, when making an adhesive, the hardener is often added from a drop bottle into a measured amount of resin which is then mixed and applied to small articles by hand. The "pot life" depends upon the hardener and the temperature. Any skin irritation from the system is most likely to occur during the active "pot life". In the case of castings, glass fibre laminates or potting of electrical compounds, the mould is first painted with a release agent, normally a silicone, but lacquers, wax polishes, wax in carbon tetrachloride or watery solutions of polyvinyl alcohol or methyl cellulose may be employed. For glass fibre laminates, the woven cloth, roving or chopped mat may be impregnated with the system, as with the polyesters, often by hand brushing, in the wet lay-up method (known in the trade as the "bucket and brush"). The plies of glass cloth, after impregnation, are laid in turn in the coated mould.

When dermatitis occurs, the epoxide-amine system should be considered as a whole; both the resins and the amines, or neither, may be at fault.

The amines were formerly considered to be the main offenders but Lea *et al.* (1958) indicate the high irritant and sensitizing capacity of the epoxy modifier included in the resin. He gives these comparative figures from patch tests; as primary irritants with 100% concentration, uncured epoxy, 48%, uncured epoxy modifier, 100% and three amine hardeners 44%, 90%, 100% positive reactors; as sensitizers, uncured resin, 20%, uncured resin modifier, 20% and three curing agents each 12% positive reactors. There is little or no risk in handling the high molecular weight solid resins; risks are greater with liquid products, especially those used without a solvent. The possible effects may be summarized as follows.

Resin.

- (i) Irritant and sensitizing effect of low molecular weight resins (Morris, 1957).
- (ii) Resin modifiers—irritant and sensitizing (Lea *et al.*, 1958).
- (iii) Unreacted epichlorhydrin—irritant.

(iv) Solvent diluents—irritant.

(v) Increase of sensitization effect of amine curing agents by low molecular weight resins (S.T.B., 1958).

Curing Agents—Ethylene Amines.

(i) Alkali effect.

(ii) Primary irritant dermatitis, caustic burns, vesicant effect and eye irritant.

(iii) Sensitization. Greater risks in short-chain amines.

(iv) Vapour—irritant and sensitizing to eyes and mucous membranes.

(v) Dye effect—yellow discolorations.

(vi) Solvent diluents—irritant.

There is a much reduced risk from amine salts. Acid anhydrides are much less toxic or irritant, though slight tingling of the skin may occur.

Cured Resin.

The cured resin should entail no risk because the ratio of hardener to resin is adjusted to avoid leaving uncombined amines, but sensitization has been reported (Bourne, 1956).

Possibilities of dermatitis exist from :

(i) Uncombined amine catalysts.

(ii) Uncombined diluents.

(iii) Solvents (acetone, toluene, xylene, methyl ethyl ketone); also when used for hand cleaning.

(iv) Accelerators—catechol—primary irritant effect.

(v) Other resins—formaldehyde based or polysulphide (Thiokol).

(vi) Plasticizers.

(vii) Traumatic effect of rubber glove fingers adhering together.

(viii) Glass fibre cuts or abrasions predisposing to sensitization.

Dermatitis usually occurs on exposed areas of hands and face. Grandjean (1957) described two groups; one with a phase lasting a week to some months before the appearance of itching and erythema which cleared on removal from contact and the worker was able to return to work. This was either a primary irritant effect or an early manifestation of sensitization. The second group, with an explosive sensitization reaction, could not return to contact.

Working conditions which increase the risk of dermatitis are the employment of unskilled workers on large jobs such as motor car bodies, ignorance of the hazards, poor hygiene and ventilation, hot humid days and sweating, contamination of skin, clothes and working benches, uncured work with a long "pot life" awaiting cure in poorly ventilated rooms and exposure to uncured resin in drilling or fettling laminates or with added trauma from glass fibre.

Attempts are in hand to use amine curing agents of low irritation potential by introduction of selected functional groups which do not interfere with the

amine cure (Godard *et al.*, 1958). It is obvious that the epoxy-amine adhesive system has some potential for irritant and sensitization dermatitis, but as experience of its use grows it is clear that with a proper awareness of the hazards and intelligent use, there seems no reason why these risks should not be properly contained or avoided.

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THE SKIN EFFECTS OF PLASTICS.*

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ONE of the most notable group of products made by the chemical industry in this century is that included under the general name "plastics". The large number of plastics now available and the extraordinary possibilities of future developments are due almost entirely to the ability of the modern chemist to synthesize the basic ingredient that is common to all of them. This is the polymerized chemical more commonly known as the polymer which in its simplest form consists of identical molecules joined together in a straight chain, e.g. polythene. The polymer is always an inert solid but made from active liquids or gases known as monomers and these chemicals may be irritants, sensitizers, corrosives, vesicants, narcotics or have some other noxious property. It must be stressed however that on the other hand monomers can be comparatively harmless e.g. ethylene, propylene, methyl methacrylate, butadiene.

In the first place, it is the factory worker engaged in manufacturing plastic raw materials and later in handling or fabricating them that is exposed to the effects of these monomers and other chemicals used in manufacture or added to influence the property of the final plastic. The term plastic raw materials may be somewhat confusing since it does not imply the basic chemicals from which they are made but is intended to describe glues, "resins" and moulding powders that are made by the larger chemical organizations. These raw materials are subsequently distributed to hundreds of smaller factories throughout the country where the resins and moulding powders are then injection or compression moulded, extruded, laminated, vacuum formed, shaped or fabricated by these or other techniques to make the final plastic article.

The highest incidence of dermatitis associated with plastics occurs in these smaller factories making the final article and is due to handling incompletely polymerized substances, chemicals such as solvents used in the process and far less commonly other chemicals added to the polymer. The skin reactions of many of these added chemicals have been investigated and described briefly by Mallette and Haam (1952*a* and *b*) although in such high concentrations (25-100%) that the results are chiefly of value to the Industrial Medical Officer in his consideration of the factory worker who may handle them in their pure state. Many of them are not skin irritants or are used in such small quantities

* Based on a paper read at a Symposium on Newer Dermatological Hazards in Industry held by the Section of Dermatology of the Royal Society of Medicine on 19 March 1959.

that they can be safely handled by the men taking only the precaution of wearing rubber gloves. Some of them and in particular plasticizers and catalysts have been unjustifiably condemned for their possible irritant action on the skin. Plasticizers are added to very few polymers manufactured today and catalysts are used in extremely small quantities in the earliest stages of manufacturing the raw material and in the majority of instances are completely destroyed in the process which is often totally enclosed.

The incidence of dermatitis in these factories obviously varies widely with the degree of mechanization or enclosure of the process, the ventilation and other factors influencing the working conditions. In some of the worst plants the working conditions may be surprisingly primitive and partially polymerized glues, resins and other chemicals including solvents may be used to make the final plastic article sometimes by hand while the poor ventilation allows dust and fumes to contaminate the man and his environment. It is not surprising therefore that the workmen's skin may be affected in such instances and the resulting disease differs in no way from that commonly associated with occupational dermatitis.

The experience of the largest single manufacturer of plastic raw materials in the British Commonwealth suggests that dermatitis arising from contact with monomers and added chemicals is a minor problem. In this particular industry during the last three years, 15 cases of dermatitis occurred among a total working population of over 3,000 men and almost all these cases occurred in plants making formaldehyde products. None of these men lost time or was obliged to have a permanent change of employment and this is in spite of an increasing production each year.

So much for the factory worker, but there has been an increasing tendency to regard plastics as possible irritants to the general public who use them. It has already been stated that the polymer and the final plastic article are inert. Some modern polymers for example are so inert that they can be attacked only by molten sodium or potassium and are unaffected by such chemicals as strong acids or alkalis. It is difficult to extract anything from the final plastic material unless polymerization is incomplete or unless it contains a plasticizer, which incidentally is often not a skin irritant. Many modern plastics contain few additives and no plasticizers and examples of such materials are polythene, rigid polyvinyl chloride, polymethyl methacrylate, polystyrene, nylon, butadiene and organic fluorine polymers. Many investigators support my contention that the final polymerized chemical is inert. Schwartz (1945) states that the final completely polymerized resin is seldom a cause of dermatitis and adds that exceptional cases are often due to incomplete or imperfect polymerization. Morris (1952) in a review of "condensation plastics" states that the completely polymerized finished plastics are dermatologically inert. In a detailed account of epoxy resins, Hine and his colleagues (1958) found no evidence of sensitiza-

tion in 205 volunteers patch tested with fabrics treated with cured resins. Zapp (1957) states that "our results from patch testing 209 volunteers suggest that no dermatitis hazard should result from ordinary handling and skin contact with polyurethane foam plastic". Calnan, Marten and Wilson (1958) estimated that 26 of their recorded 27 cases of hair net dermatitis were due to the dye and not to the nylon.

Thomson (1958) states that "although certain external agents cause a reaction more commonly than do others, yet in fact it is probably true to say that any substance is a potential irritant to someone somewhere". In spite of this, the practical evidence of the dermatological inertness of plastics is shown by the use of nylon stockings, socks and gloves, in close contact with parts of the body frequently affected by contact or occupational dermatitis, clothing and underclothing made of synthetic fibres, kitchenware, telephones and fountain pens by millions of people with only a minute percentage of reactions. Twenty years ago, Great Britain manufactured 30,000 tons of plastic raw materials. Last year it produced more than 400,000 tons whilst West Germany produced half as much again and the United States five times this quantity. In the same year, the largest single manufacturer in this country using active chemicals in making a wide variety of different plastic products reported only two cases of dermatitis.

It is obviously possible in this short space to consider the skin effects of plastics only in this very general fashion but I hope that my paper suggests that a sense of proportion is an important asset to anyone considering this problem in its widest implications.

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CEMENT AND OIL DERMATITIS. THE PART PLAYED BY CHROME SENSITIVITY.

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PIRILÄ AND KILPIO (1949) reported that ten sufferers from cement dermatitis were sensitive to chrome, and Jaeger and Pelloni (1950) recorded that 94% of 32 cases had positive patch tests to potassium dichromate. These investigations started off a completely new line of thought on cement dermatitis. Jaeger and Pelloni also showed that samples of cement contained minute quantities of chromium and they considered these two findings were complementary.

Following this discovery, reports of chrome sensitivity in cement workers were made in several European countries. Frey (1952) tested a series of 33 cases of cement dermatitis and found that they were all hypersensitive to potassium dichromate. At the same time a control series of 33 other cases of industrial dermatitis produced only 2 positive results. Engelbrigsten (1952) described a cement manufacturing plant where dermatitis was rare. However, he tested eight cases and found them all to be sensitive to chrome and he also demonstrated that all the samples of cement he tested contained chromium. Similar reports were published in 1952 by Spier and Natzel and by Engelbrecht.

In 1953 this subject was discussed in papers by Pontes de Carvalho and Brum Negreiros, Skog and Thyresson and Sidi and Longueville. Pirilä (1954) published a large series of 90 patients, 74 of whom showed positive patch tests with 0.5% potassium dichromate, while in a series of 51 cases of other types of eczema no positive results were elicited. Denton *et al.* (1954) discussed the soluble compounds of chromium present in cement.

No reports have been found in the literature stating that chrome sensitivity has been directly related to exposure to oil. Winston and Walsh (1951) reported positive patch tests in railroad workers but they had worked with oil which had been treated with potassium dichromate as an anti-rust agent. Similar reports, in the same industry, were made by Guy (1954).

The following report is of work designed to investigate the part played by chrome sensitivity in cement and oil dermatitis in Northern Ireland. Most of the patients involved in this study were referred by the dermatologist who saw them in regard to their claims for Industrial Injuries Benefit. Patients were patch tested with chromium solutions as were a control series of patients with other types of eczema. A spectrographic search was made for chromium in various samples of cement and oil and where present the amount was estimated colorimetrically.

PATCH TESTS WITH POTASSIUM DICHROMATE.

All patients were first patch tested with potassium dichromate. These were not necessarily recent cases but had been so diagnosed over a period of three years. To avoid false positive results or generalization of the reaction, no patient who had an acute dermatitis was patch tested at that stage. A total of 200 patch tests with potassium dichromate was carried out, 134 patients suffering from oil dermatitis and the remaining 66 from cement dermatitis.

It was thought important that a series of control tests should be done with potassium dichromate. Bonnevie (1939) reported that 5.5% of his eczema patients reacted positively to patch tests with this compound. Schwartz *et al.* (1947) and Engelhardt and Mayer (1949) also discussed the finding of chrome sensitivity in eczema patients. For this reason it was decided to patch test a sample of the cases of eczema and dermatitis encountered routinely in the dermatological out-patient department and not caused either by cement or oil. This series of control cases numbered 102 and was made up as shown in Table I.

TABLE I.—*The Number of Cases of Each Type of Eczema/Dermatitis Used in the Control Series.*

Type of eczema.					Number.
Cheiopompholyx	.	.	.	.	20
Contact eczema :	soaps	8	}	.	18
	resins	8			
	elastoplast	2			
Nummular eczema	.	.	.	.	16
Seborrhoeic eczema	.	.	.	.	16
Hypostatic eczema	.	.	.	.	15
Flexural eczema	.	.	.	.	12
Light sensitivity	.	.	.	.	5
Total	.	.	.	.	102

Technique of Patch Testing.

The test substance was applied to the skin on a double piece of lint 1 inch square. This was covered by a piece of "Sellotape" $1\frac{1}{2}$ inches square and these were attached to the skin using a rectangular piece of zinc oxide plaster measuring 2 inches by 4 inches. In all cases the site of application was the outer surface of the upper arm. The lint was soaked in the test substance and then firmly pressed until there was no further dripping. This was important because the amount of substance used as well as its concentration may have some effect on the reaction caused (Pirilä and Kilpio, 1949).

The tests were normally applied for 48 hours, removed after that time and the results read. Every patient was instructed that he should remove the test from the skin should there be evidence of excessive weeping or should there be pain. He was also warned that there might be quite severe itching. In fact a few patients with cement dermatitis did remove the test. The patients were not seen again after the first reading but those who showed a negative result were instructed to report if any reaction occurred after the 48-hour period. This was thought important because it has been reported that 10% of positive tests are delayed beyond the first 48 hours but

become positive within the first five days (Calnan, 1955). Positive results appearing after the first five days are said by Sulzberger (1940) to be due to a different mechanism. However, in no instance did a patient report such an event in this series.

Strength of Potassium Dichromate.

Bonnevie when patch-testing with potassium dichromate used a 0.5% solution. This had also been the concentration used by Engelhardt and Mayer (1929) and by Bering and Zitzke (1935). Probably because of this, Jaeger and Pelloni also tested with a 0.5% solution when they made the discovery of chrome sensitivity in cement workers. In fact in most of the series of tests published on cement dermatitis 0.5% potassium dichromate was the test substance. However, Pirilä states that patients may be sensitive to a 0.1% solution and Jaeger and Pelloni also mention this fact.

At first in this work 0.5% potassium dichromate was used. It was very soon obvious that this solution was too strong so that a 0.1% strength was then used routinely.

The Reading of Results.

The most difficult part of patch-testing lies in the interpretation of the results, and it is true to say that this method of testing has, to some extent, lost favour because of the many pitfalls. The real problem is to distinguish an allergic reaction from one which is a primary irritant. A neat vesicular reaction is normally taken as allergic while a grossly oedematous one with a bulla is almost sure to be due to primary irritation. In between, however, there are many more types of reaction which can prove very difficult to interpret.

Sulzberger and Witten (1953) wrote the following opinion concerning the reading of patch tests. "While the clinical appearance of a primary irritant response and an allergic eczematous response are not always markedly different, the presence of closely-set clear vesicles favours the assumption of the allergic reaction while minute papules and pustulo-vesicles irregularly situated at follicular or sweat gland orifices, as well as follicular crusts and minute superficial etched-out ulcers, are more common in primary irritant reactions."

Another serious source of error in the reading of patch tests is the false positive reaction. This reaction is more difficult to differentiate from the true allergic response than a toxic one. For this reason the positive patch test must be estimated very cautiously in the presence of extensive or acute eczema.

RESULTS.

Cement dermatitis.—Sixty-six sufferers from cement dermatitis were tested. Of this number 63 were taken to have reacted positively and three negatively. In most cases there was a collection of vesicles under the whole area of the patch, often accompanied by moderate weeping. It was noted that in several instances the reaction spread over a small area beyond the limits of the piece of lint. In a few cases the reaction consisted of redness and papule formation with a few vesicles. In the case of the cement tests the positive results did not give rise to serious doubts about the reaction being primary irritant but, as will be seen, there was slightly more difficulty in the oil cases.

The three patients who reacted negatively showed absolutely no response to the patch test. Of the three, two had a fairly widespread dermatitis while

the other had only involvement of the hands. Extra care was taken with these three patients to make sure no delayed positive result had occurred.

Oil Dermatitis.—One hundred and thirty-four patients whose dermatitis was attributable to oil were investigated. This group did not include any cases of oil folliculitis. The sufferers from oil dermatitis were engaged in many different occupations and used many different types of oil. There were two clear cut groups of workers and a miscellaneous third group. These groups were 49 mill workers—mostly women, 13 men using cutting and cooling oils and 72 others, who worked with various oils including light and heavy fuel oils, lubricating oils and heavy grease.

In this series of tests, different types of reaction appeared. A common finding was of irregularly situated little papules without any redness or vesicle formation. In a few instances little pustules appeared which gave the impression of blocked sweat glands or hair follicles. These reactions by no means involved the whole area covered by the patch but occupied a small area only. It was thought that these findings were due to the primary irritant action of the potassium dichromate. Twenty-one of the oil patients showed this result. In eight cases, positive results were obtained in every way comparable to those in cement workers. The remaining tests caused no reaction whatsoever in the skin.

Controls.—As stated earlier, 102 eczema patients were patch tested as controls. In none of the cases was a true allergic reaction to the bichromate elicited. This finding does not agree with the results of Bonnevie (1939) or with those of Engelhardt and Mayer (1949). On the other hand Pirilä (1954) studied 51 cases of eczema and found none of these to be chrome sensitive. Thirteen of my patients produced a primary irritant reaction.

TESTS WITH OTHER COMPOUNDS OF CHROMIUM.

In all the patch tests described so far the chromium compound used was the anionic hexavalent salt. It was thought that it would be of interest to discover whether or not patients hypersensitive to this chromium salt were also hypersensitive to some of the other salts. However, due to the fact that most of the other chromium compounds were either corrosive or insoluble, the trivalent cationic salt, chromium chloride, was the only one thought suitable for patch testing.

A solution of chromium chloride with the same molarity as 0.1% potassium dichromate was prepared and the patch test was performed in exactly the same way as previously. Nineteen of those patients hypersensitive to potassium dichromate were thus tested and 14 showed negative results, 4 a primary irritant type of reaction, while the other case produced a reaction indistinguishable from a true allergic one. This was in a patient with a very severe dermatitis which had caused frequent admissions to hospital but which was inactive

at the time of the test. It was thought, in view of the other negative results, that this could possibly be a false positive due to the patient's very reactive skin.

THRESHOLD OF SENSITIVITY TO POTASSIUM DICHROMATE.

The reactions obtained from using the 0.1% potassium dichromate in most of the cement workers were quite severe. A small number, for this reason, was tested with weaker solutions of the same substance. Fifteen patients were thus tested and the strength of two potassium dichromate solutions used was 0.05% and 0.01%.

In 13 of the 15 tested with 0.05% solution there was a positive result. This consisted of redness and papules with a variable number of vesicles and the reaction covered the whole area of skin touched by the patch. The other two patients showed very little skin change and certainly could not have been confidently stated to be positive.

The same 15 men were tested with the 0.01% solution. Of these, 5 produced a reaction which could fairly safely be called positive. It consisted of slight swelling and redness under the lint with a variable amount of papules and vesicles. The other ten showed no reaction.

SEARCH FOR CHROMIUM.

As stated in the introduction, various samples of cement and oil were tested for the presence of chromium and when it was found, the amount was estimated colorimetrically.

METHODS.

(i) Spectrography.

1. *Cement*.—Using samples containing diminishing quantities of chromium it was found possible to detect one part per million of chromium using the Hilger medium quartz spectrograph. Loss by spurring from the electrode, however, made this low level difficult to reproduce. The cement sample was mixed with its own weight of carbon before packing, the graphite rod being used as the electrode. Using the medium quartz spectrograph it was seen that the separation of the lines was not very great so that recognition of the correct lines was difficult. The large dispersion spectrograph was therefore used and all cement samples tested showed easily distinguishable chromium lines.

2. *Oils*.—Oils were collected from as many sources as possible after taking a detailed industrial history from the sufferers from oil dermatitis. For spectrographic examination of oils three methods were used.

(a) Soaking of electrodes.—This method was described by Carlson and Gunn (1950). No chromium lines were obtained from any oils by this method.

(b) Ashing of oil.—This method was described by Karchmer and Gunn (1952) and Gunn and Powers (1952). Again, no chromium lines were discovered by this method.

(c) Concentration technique.—This method was described by Mitchell (1956). Traces of chromium were discovered in several oils by this method but it was thought possible that these traces might have been introduced as impurities from the many

reagents used in this method. This possibility was strengthened by the fact that blanks carried out with the reagents gave a slight colour reaction with diphenylcarbazide.

(ii) *Colorimetric Determination of Chromium.*

Small amounts of chromium may be determined colorimetrically using sym-di-phenylcarbazide reagent in mineral acid solutions. The chromium must be in the hexavalent form to give the correct colour.

Many methods were tried with this reagent to determine the amount of chromium in samples of cement and the final one was as follows. One g. of cement was fused with 4 g. of sodium carbonate in a platinum crucible. After the silica was removed the solution was then made definitely alkaline with sodium hydroxide and 3 ml. of bromine water added. This was heated on the water bath for thirty minutes. The heavy precipitate containing iron was then removed by centrifuging. The clear supernatant fluid from the centrifuge was acidified with 10 N sulphuric acid so that the final 100 ml. solution would be 0.25–0.3 N. One drop of phenol served to remove excess bromine. Two ml. of orthophosphoric acid were used to stabilize the colour and the solution made up to 100 ml. with distilled water and 1 ml. of diphenylcarbazide. The colour was read after ten minutes using the 1 cm. cell in the EEL absorptiometer with the 605 filter. The results are shown in Table II.

TABLE II.—*Total Chromium Present in 12 Samples of Portland Cement.*

Portland cement sample number.	Total chromium in $\mu\text{g./g.}$
1	65
2	51
3	44
4	52
5	58
6	46
7	49
8	61
9	53
10	46
11	54
12	50

Probably of greater interest than the total chromium is the amount of soluble hexavalent chromium in the different samples. To estimate this 5 g. of cement were shaken quite vigorously for three hours in approximately 75 ml. of distilled water. After filtering through a Whatman No. 1 filter paper on a Buchner funnel the clear filtrate was acidified with 10 N sulphuric acid. This was again done using diphenylcarbazide and the following results obtained.

TABLE III.—*Soluble Hexavalent Chromium Present in 12 Samples of Portland Cement.*

Portland cement sample number.	Soluble hexavalent chromium in $\mu\text{g./g.}$
1	3.9
2	3.1
3	3.0
4	3.7
5	3.7
6	4.4
7	4.4
8	4.2
9	4.25
10	4.8
11	3.9
12	3.3

Attempts were made on several oil samples to produce colour representing chromium using diphenylcarbazide, but without success. Following this it was seen possible to recover 80% of chromium added to oil samples so it seemed safe to assume that the oils contained no appreciable chromium.

DISCUSSION.

Cement Dermatitis.

In the present series of cases of cement dermatitis 95.4% of patients reacted positively to a patch test with potassium dichromate. At the same time 18 of 19 of these positive cases showed a negative test with chromium chloride. It has also been shown that cements manufactured and used in Northern Ireland contain chromium and indeed they contain chromium in the soluble hexavalent form. It must be assumed, then, in view of the large percentage of positive results, that chrome sensitivity has a large part to play in cement dermatitis. However, it cannot be considered that this condition is a simple one of chrome allergy.

Jaeger (1955) stated that he considered the various physical factors, which were thought to be the full cause of the dermatitis, still had importance in the aetiology. He considered that it was probable that such factors as alkalinity, water, sweat and abrasions in some way "opened the gates to sensitization to the salts of chrome."

Keil (1945) discussed factors which predisposed to cutaneous sensitization to any simple chemical substance. He mentioned friction, maceration, breaks in the continuity of the skin, heat and perspiration, as important factors—presumably their action is to increase the degree of contact of the allergen with the skin.

Attempts to find the threshold of sensitivity to chromates in this work seem to indicate, that the 0.1% solution is the most dilute which gives constant results. A few patients reacted weakly to the 0.01% solution. In view of this it is interesting to note that Jaeger and Pelloni (1950) stated that 18 of their 34 cases gave a positive patch test with cement. Other authors have also observed that cements which elicit a positive patch test contain considerably less chromates than the lowest concentration of a chromate solution which will show a similar reaction on the skin. As will be seen the usual concentration of soluble hexavalent chromium salts in cement is of the order of 3 or 4 $\mu\text{g./g.}$ This strange happening might be explained by postulating that chromates can exert a stronger action in the alkaline *milieu* of the cement. Spier and Natzel (1953) were able to show that chromates dissolved in a buffer of pH 10.3 may produce a positive reaction at considerably lower concentrations than if the same amount of chromate was dissolved in water.

Quite apart from these patch tests with cement, it may be thought strange that cutaneous hypersensitivity to chromates could develop from contact

with cement which contains only 3 or 4 parts per million of hexavalent chromium. In the case, however, of the man working continually with cement, all the physical factors previously mentioned come into play. Pirilä (1954) discusses a similar phenomenon which occurs in the pottery industry in which small amounts of cobalt are added to the mass of clay. In many cases of eczema due to sensitization to cobalt in this industry the amount of the element in the mass is usually insufficient to produce a positive patch test reaction. Then, as pointed out by Denton *et al.* (1954), moisture evaporates from cement slurries in contact with the skin so that the percentage of hexavalent chromium rises. When the soluble chromates from cement go into solution in the worker's sweat considerably stronger solutions of the salt may be formed. All these factors tend to increase the amount of chromium making contact with the skin.

In this series there were three patients who suffered from cement dermatitis but did not show cutaneous hypersensitivity to the bichromate. Each was repeatedly questioned about his work and leisure hours and it was thought that in each the correct diagnosis had been reached.

Oil Dermatitis

In approximately 6% of the 134 patients with oil dermatitis a positive patch test was elicited. It would at first appear that these were coincidental results, agreeing, for instance, with the eczema material of Bonnevie. However, the occupations of these eight men seemed significant. Six were employed on ships as donkey-greasers and it was found that their occupation was concerned with oiling and cleaning the engines and between times painting parts of the ship with anti-rust agents. The other two men were a body-builder's helper (repairing coachwork on buses, etc.) and a sheet-metal worker. It was found that these two were also, at times, engaged in treating metals with rust preventive. In view of the inability to show that oil contained significant amounts of chromium it was thought that these men may have come into contact with the element elsewhere at their work and the anti-rust solutions were therefore tested spectrographically. One was "red lead" and the other a proprietary product ("Metagalv"). Both contained appreciable amounts of chromium. "Metagalv" contained very great amounts of zinc and chromium and seemed to be similar to the old zinc-chromate primer which has in the past caused chrome dermatitis. This was one possible source of the chromium and no doubt there may have been other sources in the occupations of these men. The heavy oils used by these workers could easily have prepared their skin for the entrance of chromium, for example, by removing the surface lipids. It was considered quite likely that the chromium sensitivity seen in these patients resulted from chromates encountered at their work but not necessarily in the oils.

SUMMARY.

In a series of 66 patients with cement dermatitis 95.4% reacted positively to patch tests with 0.1% potassium dichromate. No positive reactions to the same test occurred in 102 patients with eczema from other causes. The weakest solution of potassium dichromate to show consistent results was 0.1% although many patients reacted to a 0.05% solution and some to a 0.01% solution. Nineteen patients whose skin was hypersensitive to potassium dichromate were tested with a solution of chromium chloride. Eighteen of these showed a negative response while the other patient had a vesicular reaction which may have been a false positive.

Spectrographic analysis of cement manufactured in Northern Ireland showed chromium to be present in all the samples tested. Using colorimetric techniques the amount of chromium was measured in 18 samples of local cement and the soluble hexavalent chromium content was seen to be in the order of 3 to 4 $\mu\text{g./g.}$ of cement.

In a series of 134 patients with oil dermatitis, only 8 (6%) showed positive patch tests with 0.1% potassium dichromate. Six of the eight positive reactors were donkey-greasers on ships when they developed dermatitis.

In view of the finding that there was very little, if any, chromium in oils, (even those oils taken from the engines of ships) it was especially interesting that the six donkey-greasers should be chrome-sensitive. These men may have encountered chromium somewhere else in their occupations. Red lead and "Metagalv", two common anti-rust agents, were tested and both were shown to contain chromium. As ships' donkey-greasers spend a large proportion of their time using anti-rust agents it was considered that these substances were a possible source of chromium. The heavy dirty oils encountered on ships possibly could prepare the skin for sensitization in a similar way to cement.

I wish to record my thanks to Dr. Martin Beare for suggesting this work and for his continued advice and help. Mr. S. McConaghy took an interest in the spectrographic and analytical work.

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ROYAL SOCIETY OF MEDICINE.

SECTION OF DERMATOLOGY.

President—Prof. JOHN T. INGRAM.

19 March 1959.

A symposium on The Newer Dermatological Hazards of Industry was introduced by Dr. Sibyl Horner. Dr. G. Hodgson spoke on Some New Adhesives and Bonding Materials ; Dr. D. K. Harris on The Skin Effects of Plastics and the late Dr. A. J. M. Johnston on Constitutional and Extrinsic Factors in Cement Dermatitis. The first three papers are published elsewhere in this issue. Dr. Johnston's work has appeared in *Trans. St. John's Hosp. derm., Soc. Lond.*, 1958, **41**, 11.

Occupational Argiria.—Dr. R. J. CAIRNS.

A man aged 56, a process metal refiner.

In 1933 he began work manufacturing silver nitrate by a process involving the immersion of silver ingots in nitric acid. Although there was no contamination of the skin through splashing, there was considerable exposure to fumes. He and the other workers in the same department became progressively darker and this was more noticeable after exposure to sunlight.

In 1955 he was referred to hospital because of hoarseness of the voice and blood-stained sputum. A bronchoscopy carried out by Mr. J. P. Lipscombe revealed the mucous membrane of the nose and bronchi to be considerably stained. The oro pharynx and larynx were clear. It was noticed that the exposed parts of the skin and the conjunctivae were heavily pigmented. There was no evidence of any new growth. There has been no exposure to silver for the past two years.

Clinical findings.—On examination now the palpebral and bulbar conjunctivae are brownish-grey. The face, hands and arms still show a faint blue-grey discoloration.

Investigations.

Histology (Dr. H. Haber).—The following sites were biopsied and sections examined for the presence of silver:

Septum: right + left +.

Right inferior turbinate: anterior end + posterior end +.

Bronchus: left lower lobe + right lower lobe + right upper lobe +.

Carena +. Skin: abdomen — forehead +.

Comments.

Although no cause has been found for the several attacks of hoarseness and blood-stained sputum, it seems unlikely that these symptoms were due to argyria of the upper respiratory tract. These symptoms, however, led to investigation which revealed silver staining of the upper air passages. This staining was most intense at the sites where eddy currents and a certain amount of stasis might occur. The cutaneous pigmentation has faded considerably over the last two years, but the distribution can still be seen clearly under Wood's light.

(Case shown by the courtesy of Mr. J. P. Lipscombe.)

Dr. C. H. WHITTLE : How did the stuff get in, did he swallow it ? Silver is not very volatile.
 Dr. CAIRNS : There appears to be a suspension of silver or silver salt in the air over the vat. This can be seen in bright sunshine as a kind of dust arising from the vat. I regard the pigmentation of the upper respiratory tract as evidence of inhalation. The cutaneous distribution corresponds most accurately to the light exposed areas of his skin and probably light has been an important factor in determining the degree of cutaneous pigmentation.

The following cases have been reported in *Proc. R. Soc. Med.* 52, 847.

Melanosis of Face from "Cutting" Oil.—Dr. R. P. WARIN.

Creosote Melanosis with Associated Vitiligo and Depigmentation Due to Monobenzyl-hydroquinone.—Dr. P. HALL-SMITH.

Angio-endothelioma.—Dr. STEPHEN GOLD.

"Neurotrophic Atrophy"—Dr. J. SCHNEEWEISS for Dr. S. C. GOLD.

Leukoderma with Loss of Hair ? Due to Photographic Developer.—Dr. K. W. LOVEL.

The following cases were also shown :

Mercury Sensitivity in a Dental Student.—Dr. BRIAN RUSSELL.

Contact Dermatitis from Cheese Mites.—Dr. J. EVERALL. (With a note on "Food Hygiene in Retail Shops" by Dr. E. W. GIBBS.)

Dermatitis from Multiple Drugs.—Dr. J. Everall for Dr. MITCHELL-HEGGS and Dr. R. D. MOYLE.

Dermatitis from Epoxy Resin.—Dr. M. FEIWEL.

Dermatitis from Chromates.—Dr. M. Feiweil.

Guttate Morphea.—Dr. Stephen Gold.

Eczema of External Auditory Meatus to Demonstrate an Applicator for Local Therapy.—Mr. A. B. ALEXANDER.

Papulonecrotic Tuberculides.—Dr. E. WILSON JONES.

? Papulonecrotic Tuberculides, Tuberculous Lymphadenopathy.—Dr. Z. CHAMBERLAIN for Dr. M. FEIWEL.

Neurodermatitis Alba Gigantea.—Dr. H. Haber.

16 April 1959.

Nail Changes Due to Lichen Planus.—Dr. P. D. SAMMAN.

1. A woman aged 56, a cashier.

History.—This lady first had symptoms of lichen planus in September 1951 and was seen in December that year. Within the next few weeks the condition became very severe and almost universal and the mucous membranes were much involved.

By March 1952 she had noted changes in her toe nails Fig. 1 and thought her great toe nails were about to be shed. All toe nails were more opaque than normal. Around the great toe nails and the right middle toe nail there were ill defined lichen planus lesions. By the end of July 1952 the left great toe nail had been lost and a few months later the right great toe nail and the right middle toe nails were also shed. Later there was partial regrowth of the two great toe nails. The skin lesions had cleared by September 1952.

Present condition.—The left great toe nail has regrown but is much deformed Fig. 2. The right great toe nail shows partial regrowth from the inner edge of the nail fold and from the middle of the nail bed. The right middle toe nail has shown very little regrowth.

Investigations.—Examination of the nails for fungus in 1952 was negative.

Comment.—This patient shows the end result of loss of a few nails in association with severe generalized lichen planus. When the nails first became involved their bases were surrounded by lichen planus lesions.

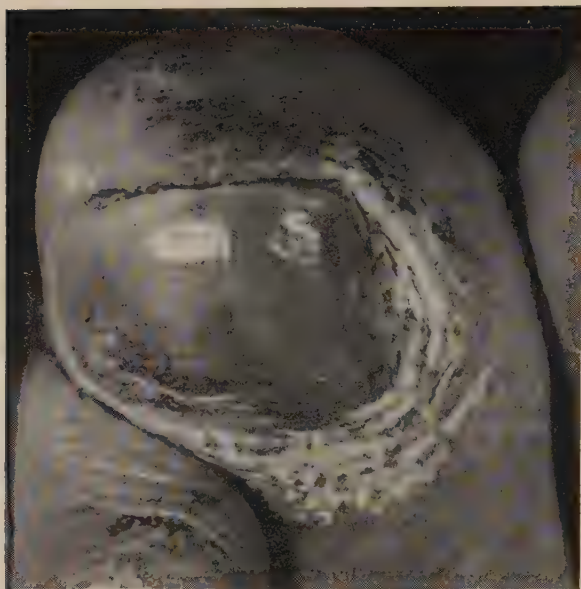


Fig. 1

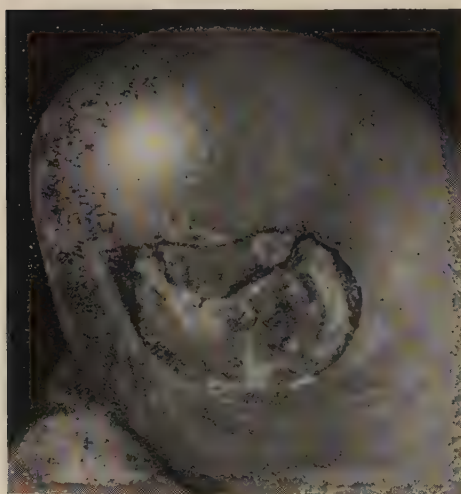


Fig. 2

A man aged 52, supervisor.

History.—This man was first seen in August 1954 with typical lichen planus papules on hands, legs and penis, and the mucous membrane in the mouth was much involved. At that time it was noted that all his finger nails were ridged. By October 1954 the nails on the ring finger of each hand had become thin and very irregular. There were no obvious lichen planus lesions around the nails. Later some of the other finger nails became involved but to a lesser extent.

Present condition.—The right ring finger nail is very thin and the eponychium has grown down as a pannus over the proximal part of the exposed nail plate.

The left ring finger nail is very thin and is curved with the finger.

All other finger nails except the left thumb are ridged and a little thinner than normal. The toe nails are not affected.

Comment.—This patient shows permanent partial destruction of two nails in connection with lichen planus. There were no obvious lichen planus papules around the affected nails at the time when the change occurred. The ridging of the other finger nails is probably unconnected with lichen planus.



Fig. 3

3. A man aged 36.

History.—This man first noticed widespread itching eruption in August 1958. He did not seek medical advice until November when he noticed that his finger nails had become rough.

Clinical findings.—When first seen on 14 November he showed widespread lichen planus papules, but it was apparent that the disease was becoming inactive. His finger nails showed thinning and ridging starting at the base and extending half way along the nail plates Fig. 3. The condition was symmetrical and all nails were involved. The distal half of the nails was normal. The toe nails appeared normal.

The skin manifestations had cleared entirely by 6 February 1959 and by then the whole of the finger nails showed thinning and longitudinal striation.

At the present time the finger nails have remained in the same condition but the two great toe nails show partial separation at their base. New nail is growing and appears to be normal.

Comment.—This patient shows what I believe to be the commonest nail change in lichen planus. The change is usually permanent but causes little disability.

THE PRESIDENT : Involvement of the hair and nails in lichen planus is important and interesting ; it differs from the involvement of nails in psoriasis, which is physiological whereas in lichen planus there is mechanical damage from infiltration. If it is persistent it causes scarring. Has Dr. Samman worked out the pathology in the nails as in the hair ?

Dr. LOUIS FORMAN : Atrophy of the hair follicles and nail matrix, and of the mucous membranes with erosions, is a serious sequel of lichen planus. When this is threatened in widespread and chronic lichen planus then steroid therapy is indicated. The dosage required is low, and the duration of treatment need be no longer than three weeks.

Dr. P. D. SAMMAN : I feel sure that steroid therapy helps some cases. At the same time, I do not think one will help the nail changes or prevent hairfall once they start ; the damage has already been done. It is possible to suppress the condition for a time but not to have any real effect on progress.

Pityriasis Rubra Pilaris (with Tylosis of Palms and Soles, and Ainhum-like Constrictions of Fingers).—Colonel P. C. MITCHELL, M.C., T.D., M.B., F.R.C.P. (Edin.), The Queen Alexandra Military Hospital, Millbank, S.W.1.

Mrs. E. S. H. Aged 32. Housewife.

History.—Not very reliable. Says that though hands were “red” in childhood there was no thickening of palms and soles. We know that by 1948 tylosis of palms and soles was present : at this time fingers were slender and scaly, but, though flexion was limited, grip was unaffected. Was able to work in a shoe shop and then in domestic service until 1956-7, when constrictions of fingers and difficulty in flexion began to appear. Recently has complained of tingling of the fingers which go white at times. Has also had scaly patches on face, back and limbs which come and go.

First pregnancy 1950, induced at 7 months—? toxæmia. Suffered from hypertension (170/120) during the second pregnancy 1957-8. Occasionally has trace of albuminuria, but renal function tests normal. Skin condition appeared to be aggravated by second pregnancy. No family history of tylosis. Two children alive and well.

Clinical findings.—Small woman with short legs and forearms. Tylosis of palms and soles with well-marked edge. Fingers atrophied and slender, and affected by an erythematous-squamous eruption. Localized constrictions affecting mainly the middle phalanges. Nails are dystrophic, but do not show changes of psoriasis. Follicular keratotic lesions on dorsum of hands. Previously there were scaly lesions on scalp, arms and back, but these are not now present.

General physical examination.—Otherwise normal.

Investigations.

Blood.—Hb. 88%. W.B.C. 5,000 per cu. mm. Poly. 75%. Eos. 4%. Lymph. 18%. Mono. 2%.

E.S.R.—16 mm./hr. *W.R. and Kahn* Negative.

Serum Calcium 9.4 mgm. %. *Serum inorganic phosphate* 2.6 mgm. %.

Urine.—Alkaline, protein—nil to trace, sugar—nil, deposit—normal.

Skiagrams.—Both hands.

“The bones of the hand are slender but their bone structure is within normal limits, apart from an impression of porosis and coarsening of trabeculae on each side of the joints. There is a moderate flexion deformity of the interphalangeal joints, particularly the distal ones. The tips of the terminal phalanges are atrophied, especially in the little finger and the right middle finger. Atrophy of soft tissue at the tips of the fingers is also visible.” (Major P. M. Bretland, R.A.M.C.)

Barium swallow.—Normal.

Histology.—Hyperkeratosis and patchy parakeratosis. The rete pegs are elongated and there is some inflammatory infiltration of the papillary processes.

Treatment.—Initially, owing to pregnancy, vitamining A was withheld. Treatment consisted of softening keratoses with salicylic acid ointment and paring with a scalpel. This improved her walking and the hand movements, but generally speaking the condition deteriorated during pregnancy.

April 1958.—Started vitamin A 600,000 units daily for six weeks with some improvement.

June 1958.—Dosage reduced to 100,000 units daily—condition deteriorated.

September 1958.—Dosage raised to 300,000 units daily for three weeks in the month, since when her condition has progressively improved.

Comment.—The association of ainhum-like constrictions of the fingers with both tylosis and pityriasis rubra pilaris (P.R.P.) is well-known. Dr. Wigley showed a case of hyperkeratosis of the palms and soles with ainhum-like constrictions of the fingers before this section in 1928. Similar cases have also been reported by Hyde and Montgomery (1900) and Acton (1927). Stelwagon (1914) has reported these constrictions in a case of P.R.P.; and in 1945 Tatz presented before this section a case similar to the one we have seen today, viz P.R.P. with keratoderma and ainhum-like constrictions of the digits.

Tatz's patient also suffered from syringomyelia, but this had supervened many years after the deformities of the hands and feet and was thought to be unconnected; though the comment that both P.R.P. and syringomyelia could be considered congenital defects was made in the discussion.

There would seem to be general agreement that these cases of constrictions of the digits, in association with tylosis and other conditions, form a group that is distinct from true ainhum in negroes. Vohwinkel has proposed for the former group the name "keratoma hereditaria mutilans"—but, in a subject already overburdened with long Latin terms, "ainhum-like constriction" would seem a simpler name.

As regards treatment, it would appear that this woman's skin improves when she takes vitamin A and deteriorates when she stops. But in a disease that is prone to natural remissions, it is very difficult to assess accurately the effects of treatment.

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Dr. J. E. M. WIGLEY: The patient to whom Colonel Mitchell has referred was shown to this Section in 1928 (*Brit. J. Derm.*, **41**, 188), but the clinical condition was entirely different from that shown today. Mine had surrounding pinkish colour; the palms and soles were like thickened pads of spongy leather, and there was also a faint yellowish tinge about them. The tips of the fingers were pointed and hard and gave an appearance of sclerodactylia. The ainhum-like constrictions consisted of fibrous tissue. When I saw the patient some few years after 1928 the other finger had been amputated.

I recall that McLeod thought that the keratoma was a typical keratoderma palmaris plantaris hereditarius and the hard fibrous tissue-like bands might have been localized bands of scleroderma.

I cannot see why the present case is not psoriasis. It has exactly the right colour. The keratoderma shown on the palms looks like psoriasis; the papules on the back do also.

The late Dr. Adamson's opinion was that psoriasis and pityriasis rubra pilaris could be interchangeable in the one patient, and he liked to refer to pityriasis rubra pilaris as a follicular psoriasis. I remember him saying that when I brought a small child to the Section of Dermatology and members agreed with the diagnosis of pityriasis rubra pilaris (*Brit. J. Derm.*, 1925, **37**, 194). They then said it had a good chance of clearing up if we did nothing. It cleared up, and three or four years later I brought the same case forward (*Brit. J. Derm.*, 1929, **40**, 274) and they then agreed the diagnosis was undoubtedly psoriasis. It would appear that Adamson's idea that the two conditions could interchange should not be left unconsidered. I think that psoriasis is the diagnosis in the case presented by Colonel Mitchell.

Dr. P. SMITH : I recently looked through the volumes of the *British Journal of Dermatology* dated 1891 to 1894. There were some dramatic illustrations of a condition described as erythema keratodes by one of the older masters than Dr. Wigley has mentioned. It was a most dramatic thing ; it appeared on the palms and soles and lasted for one or two years in one case and for six to eight years in another case. Several cases were described about that time. It seemed such an intriguing subject that I looked through subsequent literature and could not find anything like the condition. It apparently died out to appear again in the case which Colonel Mitchel has shown.

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Dr. BENTLEY-PHILLIPS : I agree with Dr. Wigley's diagnosis of psoriasis. It seems to me it might be interesting to note the effect of steroids on this patient.

THE PRESIDENT : I agree that there must be something common to the mechanism of the skin reactions in psoriasis and pityriasis rubra. I have no doubt that this patient has pityriasis rubra pilaris. I do not lay enormous emphasis on the follicular plugging.

Often pityriasis rubra pilaris is a reaction to other diseases which in older persons may be neoplastic.

I was interested that the fingers in Colonel Mitchell's case were so like acrosclerosis, but in that case atrophy arises from vascular occlusions, whereas in this case it is from external pressure.

The following cases have been reported in *Proc. R. Soc. Med.* **52**, 850.

Gynaecomastia Persisting after Oestrogen Therapy for Acne.—Dr. LOUIS FORMAN.

Kaposi's Sarcoma with a Lung Tumour.—Dr. LOUIS FORMAN.

Incontinentia Pigmenti.—Dr. P. D. SAMMAN.

Adenoid Basal Cell Epithelioma.—Mr T. HENNEBRY, Mr. V. B. LEVISON and Dr. M. FEIWEL.

The following cases were also shown :

Gout.—Dr. O. L. S. SCOTT.

Cerebello-retinal Angiomas (von Hippel-Lindau Disease).—Dr. O. L. S. SCOTT.

Porokeratosis of Mibelli.—Dr. A. AITKEN ROSS.

ANNOUNCEMENTS

TWELFTH INTERNATIONAL CONGRESS OF DERMATOLOGY, 1962.

Members of the Association may wish to be reminded that the Congress at Washington takes place during the tourist season and that enquiries about passages by sea or air should be made well in advance. A shipping company has written to the Secretary, suggesting that arrangements should be made in the near future if passages are to be guaranteed.

BRITISH ASSOCIATION OF DERMATOLOGY.

Dr. G. B. Mitchell-Heggs has received autobiographies from only a quarter of the members of the Association to whom he sent requests last year. He is anxious to complete his collection and would be grateful for replies from defaulting members in the near future.

INTERNATIONAL SOCIETY FOR TROPICAL DERMATOLOGY

DURING the Sixth International Congress for Tropical Medicine and Malaria, held in Lisbon in 1958, Professor Aldo Castellani proposed the formation of an International Society for Tropical Dermatology.

An Organizing Committee has been formed, composed of the following :

Professor Aldo Castellani, M.D., F.R.C.P., F.A.C.P., Instituto de Medicina Tropical, Lisbon, Portugal.

George Clinton Andrews, M.D., New York, N.Y., U.S.A.

Anthony C. Cipollaro, M.D., New York, N.Y., U.S.A.

Frederick Reiss, M.D., New York, N.Y., U.S.A. Organizing General Secretary.

Over 500 dermatologists, several pathologists and microbiologists in the United States and Canada have joined and about 300 abroad, representing all five continents.

The organizational meeting will be held in the near future. It has been tentatively agreed that the First International Congress of the Society be held in 1963 in Rio de Janeiro, prior to or after the International Leprosy Congress.

For further inquiries, address Dr. Frederick Reiss, 870 Fifth Avenue, New York 21, N.Y., U.S.A.

THE THIRD INTERNATIONAL CONGRESS ON PHOTOBIOLOGY.

THE Comité International de Photobiologie will hold the Third International Congress on Photobiology in Copenhagen 31 July–5 August 1960. This Congress will honour the 100th anniversary of the birth of Niels R. Finsen, who is recognized as the father of modern photo- and radiobiology, and it will be called The Finsen Memorial Congress.

The President of the International Committee on Photobiology is Dr. Alexander Hollaender, Director, Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tenn., U.S.A., the Secretary General is Prof. W. Burekhardt, Städt. Poliklinik für Dermatologie, Herman-Greulichstrasse 70, Zürich, Switzerland.

The President of The Finsen Memorial Congress is Dr. B. C. Christensen, The Finsen Memorial Hospital, Strand-boulevard 49, Copenhagen ø, Denmark. The Secretary General of the congress is Dr. B. Buchmann, Biofysisk Laboratorium, Juliane Mariesvej 30, Copenhagen ø, Denmark.

The programme of the congress will consist of contributed papers and of a series of symposia on the following topics :

Lupus vulgaris—history and treatment.

Biological action spectra.

Initial mechanisms involved in radiation effects.

Phototherapy.

Photoreceptors in aquatic organisms.

The results of the 3rd International Geophysical Year with regard to radiation.

The programme should also include lectures on the following subjects :

Niels R. Finsen and basic research.

Plant cell response to visible light excluding photosynthesis.

Biological clocks.

The effects of long visible and near infrared radiation.

Photoreactivation—invited papers.

Inquiries may be sent to the President or to the Secretary General of the congress.

The Secretary General is most anxious to be informed of the names and addresses of all persons interested in The Finsen Memorial Congress.

BOOK REVIEWS.

AN ELEMENTARY TEXTBOOK.*

WHILE this is not a book for the specialist dermatologist, teachers of dermatology might do worse than have a look at the way the subject matter is presented in this volume. After fifty pages of general considerations dealt with in a most interesting way, the authors dig into their subject proper. Dermatology is dealt with on a morphological basis and diseases are considered under chapter headings, such as macular eruptions, papular eruptions, vesicular eruptions and so on. It having been decided into which chapter each disease belongs, they are placed in alphabetical order and described in synopsis form. Lucidity is further enhanced by the employment of a wide variety of printing type. There is a lot to be said for classifying skin diseases morphologically. Numerous other systems have been tried and in the end the result has almost invariably been confusion. When it comes to etiology almost every teacher has his own ideas and it is small wonder that other branches of the profession have sometimes considered dermatologists muddle-headed.

The book is very well presented and there are numerous excellent black and white photographs. The pages are printed in two columns which is particularly restful to the reader and a great advantage while reading in a reclining position. The book is to be especially recommended to general practitioners for, in the event of their having been lucky enough to get a diagnosis from a hospital consultation, they can turn it up quickly and get at the bones of the matter, including the latest therapy, in less than two minutes. It should also prove useful to students up for their final exams.

J. L. F.

THE YEAR BOOK.†

THERE can be few who do not purchase the *Year Book* for the annual post-graduate course; unless they are in the beg, borrow or etcetera class. Like the rudimentary trunk of Rudyard Kipling's Elephant's Child deep in the Limpopo River, our intellectual processes become suddenly but painlessly extended, and the advantages of such a changed status pointed out to us as they were to him by the bi-coloured python rock snake. In his words:

'*Vantage No. 1.*—An abstract of world literature including a substantial portion on investigative studies.

'*Vantage No. 2.*—A symposium on benign pigmented lesions including the clinical examination, indications and contra-indications for biopsy, bio- and histo-chemistry and the evolution of the cellular naevi, description of the types and advice on management.

'*Vantage No. 3.*—Recent advances of treatment with both local and systemic drugs; e.g. triamcinolone, injection use of hydrocortisone, griseofulvin and trimeprazine, chelation in scleroderma with E.D.T.A. and a general leavening of the whole with the usual chapter on drug eruptions.

* *Clinical Dermatology*. HARRY M. ROBINSON JR. and RAYMOND C. V. ROBINSON (1959). London: Baillière, Tindall and Cox. Pp. 242, 117 illus. Price 68s.

† *Year Book of Dermatology and Syphilology*, 1958-59 series. R. L. BAER and V. H. WITTEN (1959) Chicago: Year Book Publishers. Pp. 477, illus, 57. Price \$8.50.

'*Vantage No. 4.*—Warnings on the hazards of just being alive in the 20th century. These describe the ill effects upon the skin of optical dyes (blankophores) and fluorescent lights, deodorants, nylon brushes, cold wave perms, multiple needles (artifact), wedding rings, lipsticks, shoes, penicillin in milk, parathion in crops, the industrial use of epoxy resins, barrier creams (primary irritants) traumatic calcinosis (miners), office duplicating papers and colour photographs with lichenoid eruptions from amines!

'*Vantage No. 5.*—A general influence for good with suggestions for standardization of terminology (so we all know what we are talking about!) e.g. benign juvenile melanoma instead of juvenile melanoma with the sinister implications of the name; Lewandowski's rosacea-like tuberculide divides into micropapular tuberculide, lupus miliaris disseminata faciei or rosacea. Systemic L.E. from hydantoin derivatives becomes L.E. like syndrome.

'*Vantage No. 6.*—Correctives on loose reasoning and conclusions reached in articles emphasized with this sort of comment, "the indiscriminate use of psychosomatic factors to account for dermatosis of unknown or relatively unknown cause!". However, some with mixed feelings may, like the Elephant's Child, say "'Scuse me" when they find that their article has been of sufficient merit to be included in the *Year Book* but still receives no comment. Others may however secretly be pleased that they have escaped but there are those lucky ones who receive the most flattering comment of all as on page 75, "We agree—Eds."! However, if one has to find faults at all in this excellent book the definition of the photographs seems to suffer by their reproduction. Also some may be disappointed that the mother elephant after such a long labour in the chapter on pigmentation gave birth to only a mouse of conclusions at the end in the way of any suggestions as to why occasional pigmented lesions in transition from junctional naevi do become malignant.

'*Vantage No. 7.*—This may be however, as many are blind and unable to appreciate a great painting until the main features have been emphasized, the conclusions above give the necessary stimulus to others to carry out research to find the answer to this problem.

To rewrite the last words of the authors' preface in summing up, "it can be said that another year has passed in which there has been much worth while reading in the *Year Book*". To those who do not do their annual reading or are in the etcetera class a further comment of the bi-coloured python rock snake is sufficient "Some people do not know what is good for them"!

G. A. H.

A WALL CHART.*

A CHEAP and accurate wall chart of the microscopical anatomy of the skin would be useful to those who teach dermatology to nurses, medical students and post-graduates. This Swedish chart, in five colours and measuring 30 inches by 40, has the merit of cheapness, although, as it is printed on paper, it would require some protection if it were not soon to become tattered. The artist has crowded in so many features that the simple skin takes on the terrifying aspect of an Hieronymus Bosch. The relative proportions of the various layers are distorted and no granular layer is shown in the epidermis. Sebaceous glands are depicted as hollow sacs lined with epithelium and the special nervous end-organs are over-stressed. The key to the features depicted contains misspellings and other inaccuracies.

* *The Skin*. Wall Chart A685. Wakefield: Education Productions Ltd. Price 7s. 6d.

CURRENT LITERATURE

THE INNERVATION OF THE HUMAN EPIDERMIS. R. P. ARTHUR and W. B. SHELLEY. (1959) *J. invest. Derm.*, **32**, 397.

These authors described their findings using an intra-vital methylene blue technique.

A. J.

A STUDY OF THE EPIDERMAL CLEAR CELLS WITH SPECIAL REFERENCE TO THEIR RELATIONSHIP TO THE CELLS OF LANGERHANS. J. FAN, R. J. SCHOENFELD and R. HUNTER. (1959) *J. invest. Derm.*, **32**, 445.

Using a gold technique these authors estimated the number of clear cells in the upper parts of guinea-pig epidermis following stimulation of the melanocytes by thorium X paintings, ultra-violet, and x-irradiations. They found a *decrease* in the Langerhans cells in the stimulated guinea-pig ear skin. They concluded that these findings supported Masson's opinion that these cells are effete melanocytes. They suggest that the stimulated melanocytes remain close to the basal layer and do not move up in the epidermis.

A. J.

NATURAL REGRESSION OF THE MELANOCYTIC NAEVUS. O. C. STEGMAIER. (1959) *J. invest. Derm.*, **32**, 413.

This worker draws attention to the natural regression of moles. This is a worthwhile contribution to the histology of naevi. It is not generally realized that these lesions can undergo spontaneous regression, and that consequently there is often a confusing histology associated with these lesions. The writer illustrates one of these difficulties in a naevus with lipomatous infiltration.

A. J.

A COMPARATIVE STUDY OF CANINE AND HUMAN DERMATOLOGY. II. CUTANEOUS TUMOURS—THE MAST CELL AND CANINE MASTOCYTOMA. M. ORKIN and R. M. SCHWARTZMAN. (1959) *J. invest. Derm.*, **32**, 451.

The authors emphasize the frequency of mast cell tumours in the dog. These lesions do not wheal on rubbing, and it is suggested that this is due to the presence of numerous eosinophils which contain antihistamine substances. They consider that there is a close similarity between dog mast cell disorders and some cases of urticaria pigmentosa; therefore these animal tumours can be used for the experimental investigations of human mast cell disease.

A. J.

ELASTOMA INTRAPAPILLARE PERFORANS (MIESCHER) NON-VERRUCIFORM AND IMITATING POROKERATOSIS OF MIBELLI. H. J. SANCHEZ CABELLERO, E. D. L. JONQUIÈRES and P. BOSQ. (1958) *Dermatologica, Basel*, **117**, 460.

The first case from the Argentine. A white boy aged 11 presented two pre-auricular plaques, that on the left being avoid and that on the right serpiginous in outline. The peripheries were slightly raised, and appeared to consist of numerous papules which had run together. The centres showed almost normal skin. Histologically, the appearances agreed with those described by Miescher, with the exception that there were no verruciform hyperkeratoses.

J. F. S.

PEMPHIGUS OR PEMPHIGOID. F. FÖLDVÁRI. (1958) *Dermatologica, Basel*, **117**, 296.

After a long preamble, in which he gives reasons for refusing to place bullous eruptions into water-tight compartments, according to such criteria as site and method of bulla-formation, acantholysis and so on. Földvári describes 20 cases in which bullae recurred on the same sites over long periods of years. The blisters were sub-epidermal for the most part: rarely subcorneal.

J. F. S.

ATROPHODERMIA VERMICULATA WITH UNUSUAL LOCALIZATION AND ASSOCIATED CONGENITAL ANOMALIES. R. KOOLJ and J. VENTER. (1959) *Dermatologica, Basel*, **118**, 161.

Atrophoderma vermiculata is a rare condition, usually confined to the face, characterized by numerous small areas of atrophy, separated by narrow ridges, giving a worm-eaten appearance.

Familial occurrence has been noted, and also its association with other congenital abnormalities. The present patient, a boy aged 18, showed lesions on his face, forearms and knees, and in addition mongolism and a congenital cardiac condition, probably Eisenmenger's complex. J. F. S.

SQUAMOUS CELL CARCINOMA ARISING IN DISCOID LUPUS ERYTHEMATOSUS. F. W. JACOBSON and H. ANNAMUNTHODO. (1958) *Dermatologica, Basel*, 117, 455.

Discoid lupus erythematosus is relatively common in Jamaica, but no instance of cancer arising on it has hitherto been reported. The present case is that of a negro woman aged 42, who had suffered from discoid i.e. of both cheeks for some six years, and complained of pain and burning in her right cheek for one year. Here there was an ulcer 3 by 4 cm. with an infiltrated base, and biopsy showed squamous carcinoma. The lesion was excised with a wide margin, and treatment with chloroquine instituted. J. F. S.

THE CLASSIFICATION OF PEMPHIGUS HAILEY-HAILEY. S. JABŁOŃSKA and T. CHORZELSKI. (1958) *Dermatologica, Basel*, 117, 24.

The authors compare six typical cases of pemphigus Hailey-Hailey with one which they consider to be bullous morbus Darier, and from clinical and cytological studies they conclude that the two conditions are quite distinct, in spite of certain resemblances. The main feature of Darier's disease is the dyskeratosis ("corps ronds" and "grains"), while in the other acantholysis predominates. A differential diagnosis can be made, too, on the basis of the clinical picture and the course of the disease. Their conclusion is that pemphigus Hailey-Hailey is an independent nosological entity. J. F. S.

RENAL HYPERPARATHYROIDISM WITH METASTATIC CALCIFICATION OF THE SKIN. T. PUTKONEN and G. A. WANGEL. (1959) *Dermatologica, Basel*, 118, 127.

This records an interesting and unusual case. A man aged 23 suffered from post-scarlatinal nephritis, with retention of phosphate, and secondary hyperparathyroidism, resulting in extensive calcareous deposits in his skin and blood-vessels. A congenital hypoplasia of his kidneys seems to have been an important factor in the development of a progressive and ultimately fatal condition. At autopsy his parathyroids were found to be greatly enlarged, and there was considerable decalcification of bone, with large collections of osteoclasts. The eruption was extensive and symmetrical, and consisted of small papules, shotty-feeling, 1 to 3 mm. in diameter, mostly of about the same colour as the normal skin, but a few brownish. Histologically, the deposits could be seen to be in the ground-substance of the connective tissue, and not in the collagenous fibres. There is a full discussion of earlier cases. J. F. S.

DARIER-ROUSSY SARCOID. S. IRGANG. (1959) *Dermatologica, Basel*, 118, 145.

A negro woman aged 60 developed a number of subcutaneous nodes on her legs, several of them after trivial injury. Irgang goes very closely into the histological picture, on the basis of which he holds that differentiation from Boeck's sarcoid is possible. The important cell is an epithelioid one, showing curious morphological and tinctorial properties, which enable one to pick it out even under a low power of the microscope. It forms miliary tubercles, without lymphocytic envelopes, and containing a few foreign-body type giant-cells. Panniculitis and vasculitis are also found, and this further marks off the Darier-Roussy from the Boeck sarcoids. J. F. S.

NEW FINDINGS ON THE EMERGENCE OF THE GROUND-SUBSTANCE IN THE CUTIS. G.-K. STEIGLEDER. (1959) *Dermatologica, Basel*, 118, 154.

A rather intricate study of a substance stainable with alcian blue and astra blue, which is released by various methods of pre-treatment of fresh normal or pathological skin. This paper is too packed with details to lend itself to summary, but is of considerable interest to histopathologists, who are recommended to read it *in extenso*. J. F. S.

DELTA-AMINO LEVULINIC ACID DETERMINATIONS IN PATIENTS WITH POLYMORPHOUS LIGHT ERUPTIONS. E. J. LEVY, M. M. CAHN, J. G. REINHOLD and B. SHAFFER. (1958). *J. invest. Derm.*, 31, 305.

Delta-amino-levulinic acid is the immediate precursor of porphobilinogen. Its ingestion has been reported to make the skin light sensitive, but 5 patients with polymorphous light eruptions didn't excrete more of it in their urine than the normal. J. H. T. D.